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Acute Epstein-Barr Virus Myocarditis Simulating Myocardial Infarction With Cardiogenic Shock

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ACUTE Epstein-Barr virus (EBV) myocarditis may yield clinical findings identical to those of acute myocardial infarction.[1-4] We describe a case of EBV myocarditis that closely resembled acute myocardial infarction complicated by cardiogenic shock.

CASE REPORT

A 38-year-old white woman went to a local hospital on Jan 7, 1986, complaining of substernal chest pain, shortness of breath, and near-syncope; an ECG was consistent with an acute anterior myocardial infarction. The patient was transferred to North Carolina Baptist Hospital for emergency cardiac catheterization.

On arrival, her systolic blood pressure was 70 mm Hg despite dopamine infusion. She appeared cyanotic, but was alert and responsive to questioning. Her oral temperature was 99.2 F (37.3 C), respiratory rate was 22/min, and pulse was thready at 150/min. Physical examination showed no jugular venous distention. Pulmonary auscultation revealed minimal bibasilar rales. Heart examination disclosed a regular tachycardia with a normal S1 and S2, and an S3 gallop. There were no murmurs. There was no hepatosplenomegaly or abdominal tenderness. The ECG showed a sinus tachycardia with 5 mm of anterior ST segment elevation and anterior Q waves (Fig 1, A). Baseline laboratory data were within normal limits except for a WBC of 16,400/cu mm with a left shift. The chest roentgenogram was normal.

On emergency cardiac catheterization, right and left coronary arteriograms were normal, but the left ventriculogram showed severe, global hypokinesia. A Swan-Ganz catheter was inserted; pulmonary artery wedge pressures averaged 24 mm Hg. The cardiac output averaged 2.5 L/min and the ejection fraction was 26%.

After catheterization, the patient was transferred to the coronary care unit for further stabilization. Additional history revealed that the patient's family had had a viral syndrome consisting of sore throat, fever, and cough during the week preceding her cardiac decompensation. Though the condition of her family members improved after several days, the patient continued to have myalgia, fever, and malaise. A severe substernal burning sensation developed, and persisted until the patient suffered the near-syncopal episode that precipitated her initial emergency room visit.

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The patient was managed in the coronary care unit from Jan 8 until Jan 15, 1986. She initially required hemodynamic monitoring, intravenous pressor therapy, and intra-aortic balloon counterpulsation to maintain her blood pressure, but her cardiac performance improved rapidly. On Jan 10, an endomyocardial biopsy showed widespread mononuclear inflammation, edema, focal fibrosis, and myocytic degeneration (Fig 2). The process appeared to be ten days old and was suggestive of a viral cause.

As the patient's cardiac performance improved, ECG tracings on Jan 8 and Jan 11 (Fig 1, B and C) showed resolution of the anterior ST segment elevation and the return of anterolateral R waves. An echocardiogram documented improvement in left ventricular performance. Cardiac isoenzyme determinations confirmed the presence of myocardial injury, with a peak creatine phosphokinase value of 573 U/L, with 12.3% MB fraction. The peak lactate dehydrogenase value was 679 U/L, with 26.9% LDH 1 and 26.3% LDH 2.

The patient had clinical improvement and became strong enough to walk. Serum was drawn for determination of convalescent viral titers on Jan 20, the 13th day after admission, and the patient was discharged. She returned on Feb 13 for repeat endomyocardial biopsy, which showed improvement, with few residual myocardial inflammatory cells and little residual fibrosis (Fig 3).

After the second biopsy, we reviewed the patient's microbiologic studies. Blood and urine cultures obtained during the acute phase of the illness remained negative for bacterial growth, and urine, stool, and throat cultures remained negative for viral growth. Acute viral titers were unrevealing. Convalescent titers for coxsackieviruses B1 through B6 and echoviruses 4, 9, 11, and 30 were $<1:8$; however, an EBV serology panel taken on Jan 20, approximately 20 days after the patient's illness began, revealed an IgG anti-EBV viral capsid antigen titer of 1:640. An anti-EBV early restricted antigen titer of 1:40 was noted, with an anti-EBV early diffuse antigen titer of $<1:10$; an anti-EBV nuclear antigen titer of 1:20 was also noted. These antibody titers indicated recent infection with the Epstein-Barr virus. There was no history of infectious mononucleosis or EBV infection. Follow-up EBV serologies done on April 24 showed a decrease in the IgG anti-EBV viral capsid antigen titer to 1:320. The anti-EBV anti-EBV early restricted antigen titer persisted at 1:40, with early diffuse titer remaining at $<1:10$; the anti-EBV nuclear antigen titer had increased to 1:40. Results of these studies were consistent with the expected immunologic response to a recent Epstein-Barr virus infection.

DISCUSSION

This patient had acute cardiovascular compromise after a viral prodrome consisting of sore

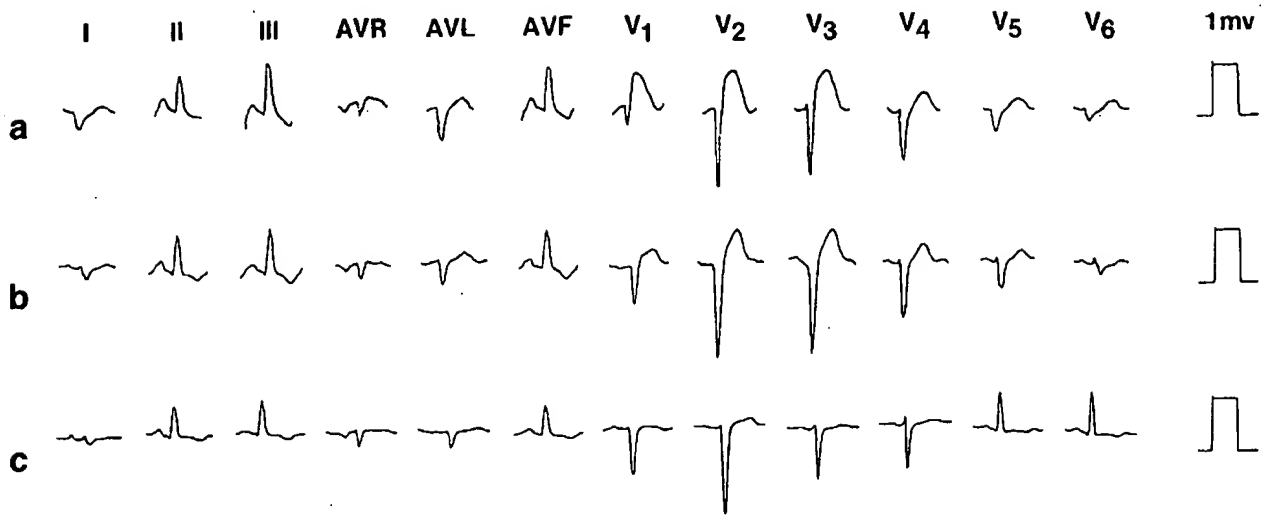

FIGURE 1. (A) Electrocardiogram at presentation on Jan 7, 1986 revealing prominent anterior ST segment elevation and pathologic Q waves. (B) Electrocardiogram Jan 11, 1986 revealing slightly lowered anterior ST segments with persistent Q waves. (C) Electrocardiogram Jan 11, 1986 revealing total resolution of anterior ST segment elevation with substantial improvement in anterolateral R wave voltage.

throat, fever, and cough. The typical findings of infectious mononucleosis, including splenomegaly, lymphadenopathy, and atypical lymphocytosis, were absent, but a specific serologic test demonstrated a recent Epstein-Barr virus infection. Most significant was the 1:40 titer of the anti-EBV early restricted antigen. Studies have shown that "anti-early antigen responses are, as a rule, transitory," and that "the mere presence of the antibody in a serum indicates, with few exceptions, a current or recent primary Epstein-Barr virus infection."[5] It seems likely that the Epstein-

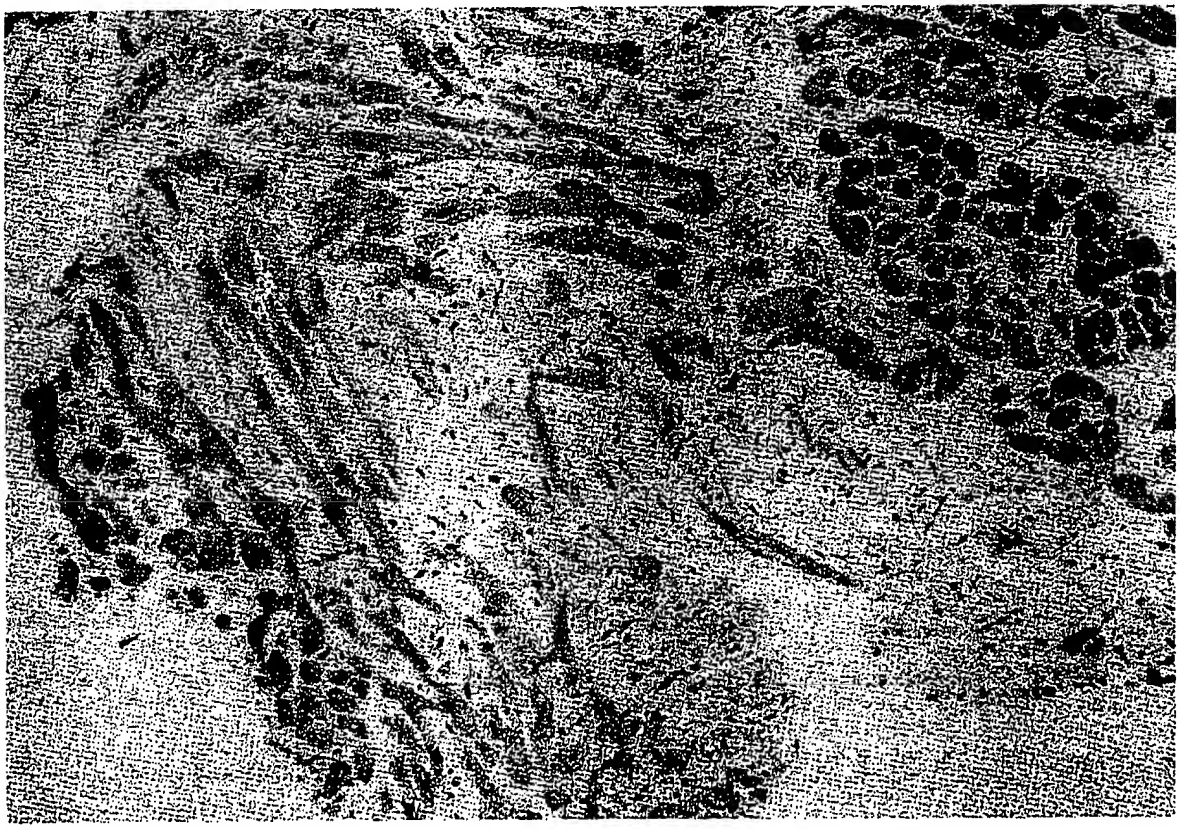

FIGURE 2. Endomyocardial biopsy on Jan 10 shows widespread mononuclear infiltration, myocytic degeneration, and focal fibrosis. (H & E, original magnification x 200)

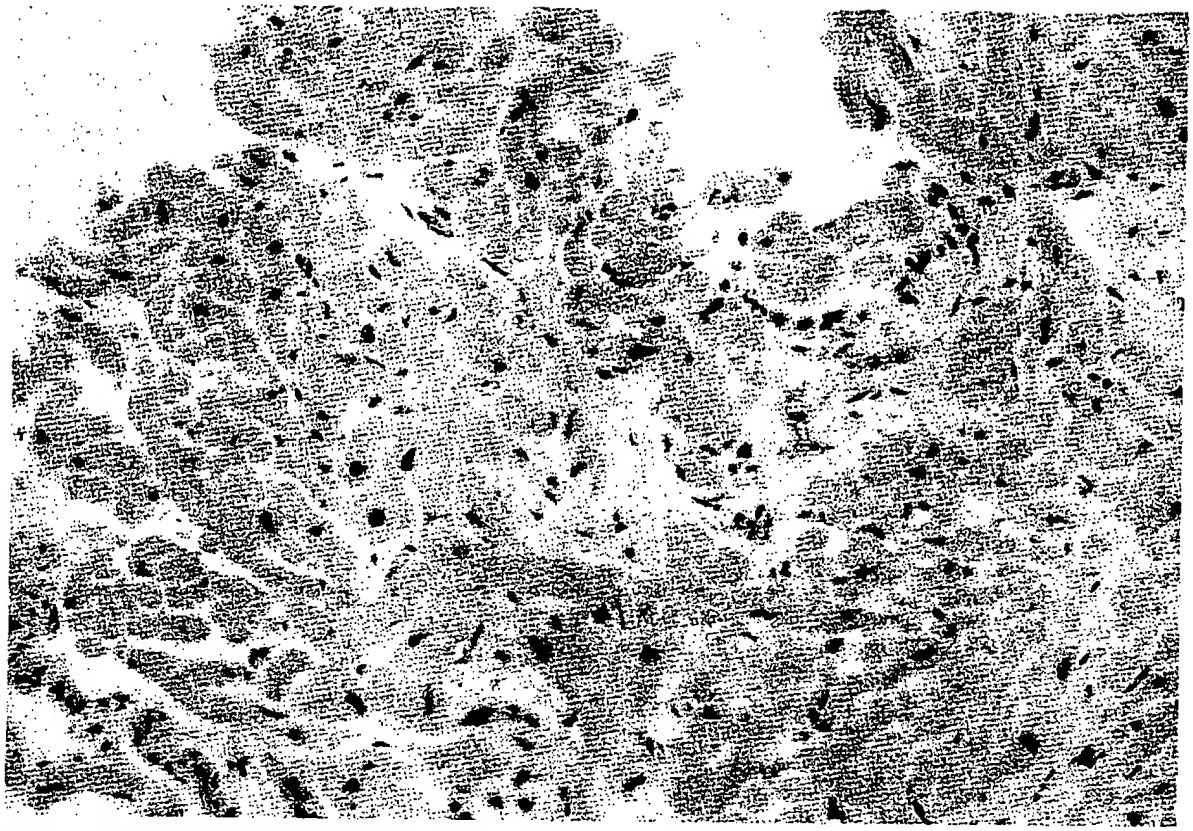

FIGURE 3. Endomyocardial biopsy on Feb 13 shows normal-appearing myocytes with little fibrosis and few residual mononuclear cells. (H & E, original magnification x 200)

Barr virus was the causative agent in this biopsy-proven case of acute myocarditis.

Initially, the clinical findings in this patient were suggestive of acute myocardial infarction. Similar cases have been reported previously. Costanzo-Nordin et al,[6] recently reported two cases of biopsy-proven myocarditis that were initially suggestive of acute infarction. More specifically, patients diagnosed as having EBV myocarditis using clinical criteria have had initial findings suggesting acute infarction. Miller et al[1] reported the case of a 17-year-old athlete who had left-sided chest pain that radiated to the left arm. Serial ECGs were consistent with an acute inferior myocardial infarction, and the heterophil titer was positive at 1:224. When cardiac catheterization showed normal coronary arteries, his illness was attributed to EBV infection. Butler et al[2] described a 29-year-old man with left-sided chest pain, myalgia, and sore throat. The ECG revealed ST segment elevation in leads II, III, aV_F, V_5, and V_6. Cardiac isoenzyme levels confirmed myocardial injury, and a diagnostic rise in the anti-EBV IgG viral capsid antigen titer was documented. The patient's illness was attributed to EBV myocarditis.

Finally, EBV myocarditis documented by biopsy has been associated with ischemic ECG changes and sudden death. Fish and Barton[3] reported the case of a 20-year-old with pleuritic chest pain and cough. The heterophil titer was 1:448 and the initial ECG revealed a new right bundle branch block with inverted T waves in leads II, III, and V_{1-4}. He suffered a cardiac arrest and could not be resuscitated. Autopsy revealed inflammatory cells in the myocardium, consistent with acute myocarditis. Frishman et al[4] described a 14-year-old girl with atypical lymphocytosis, fever, and sore throat. On the eighth day of her illness, she had cardiopulmonary arrest. Initial monitored rhythms showed ventricular fibrillation, and resuscitation was unsuccessful. Autopsy within six hours of death revealed myocardial mononuclear infiltrates, indicating recent myocarditis.

SUMMARY

We have reported the case of a 38-year-old white woman with substernal chest pain, hypotension, and ECG changes suggesting acute anterior myocardial infarction. Cardiac catheterization revealed no coronary artery pathology, but severe global hypokinesia was noted on left ventriculogram and endomyocardial biopsy revealed myocytic degeneration and mononuclear cell infiltration consistent with acute viral myocarditis.

Viral serologies confirmed a recent Epstein-Barr virus infection.

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Focal Glomerulosclerosis in Hodgkin's Disease Necessitating Peritoneal Dialysis

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THE NEPHROTIC SYNDROME has been well documented as an infrequent but clinically significant complication of Hodgkin's disease.[1-13] We report a case of focal segmental glomerulosclerosis (FGS) complicated by severe nephrotic syndrome and renal failure in a young man with Hodgkin's disease.

CASE REPORT

A 16-year-old white boy saw his personal physician in May 1983 because of a two-week history of a flu-like illness and progressive generalized edema. There was no significant past medical history. Physical examination showed blood pressure of 146/100 mm Hg, bibasilar pulmonary rales, and anasarca. Serum creatinine value, BUN value, and 24-hour urine protein excretion were 2.4 mg/dl, 43 mg/dl, and 23.5 gm, respectively; one week later, they were 4.4 mg/dl, 82 mg/dl, and 30.5 gm, respectively. Creatinine clearance was 26 ml/min.

Renal biopsy revealed findings consistent with focal segmental glomerulosclerosis. There were ten glomeruli, one of which had a small segmental area with irregular thickening of the basement membrane of the peripheral capillary loops. There were focal patchy inflammatory cell infiltrates composed predominantly of lymphocytes (Fig 1). Electron microscopy showed extensive effacement of foot processes without any electron-dense deposits. Immunofluorescence study was unremarkable.

In an attempt to control proteinuria, steroids were given (three 1 gm injections of methylprednisolone sodium succinate [Solu-Medrol], followed by 60 mg of prednisone every other day for three weeks). Hemodialysis via a percutaneous femoral catheter was required on two occasions to control uremia and intravascular volume. After one month of unsuccessful treatment, including a trial of indomethacin, a peritoneal catheter was placed and the patient was trained to perform continuous ambulatory peritoneal dialysis. The therapeutic goal was to use the dialysis to control edema and diminish proteinuria by decreasing urine volume. Figures 2 and 3 detail the daily urine volume, creatinine clearance, protein excretion, and serum albumin and globulin values. Dialysis protein excretion was 2.3 gm on one day during the seventh week of treatment. Other than occasional nausea and one episode of peritonitis due to *Staphylococcus epidermidis*, the patient tolerated dialysis without significant morbidity.

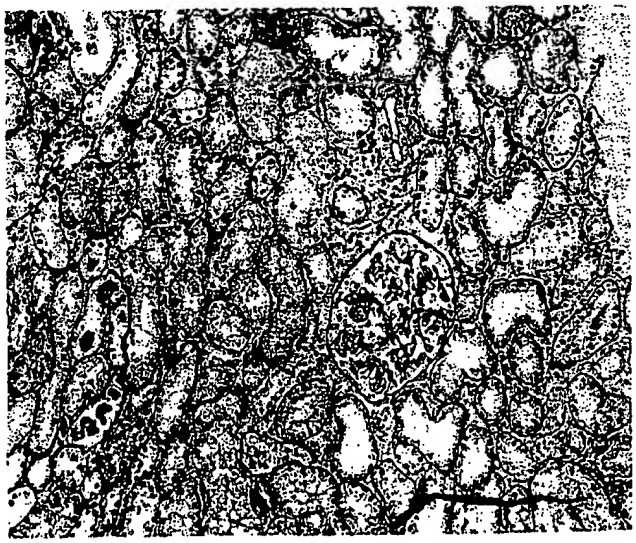

FIGURE 1. Glomerulus showing sclerotic segment. (PAM, original magnification x 100)

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